

Oesophageal hold-up of tablets taken without water has been shown radiologically and is particularly likely if gastro-oesophageal reflux or hiatus hernia is present and the patient assumes a supine posture immediately after ingestion.³ Anatomically the oesophagus is indented by the aortic arch and the left main bronchus at about 24 and 28 cm respectively from the incisor teeth. In 18 cases reviewed by Collins *et al*¹ in which the ulceration was probably due to direct contact between the tablet and mucosa and in which the site of the lesion was specified, the ulcer occurred in mid-oesophagus in 12 patients. It would seem reasonable to suggest that this site is vulnerable because of the anatomy of the area. With regard to tetracycline hydrochloride tablets in particular, the likelihood of mucosal damage is enhanced by the fact that these tablets dissolve in water to produce a highly acidic solution with pH as low as 2.3.²

We believe that the oesophageal ulceration in our patient was caused by a direct irritant effect from prolonged contact between oesophageal mucosa and a tetracycline hydrochloride tablet at about the level at which the left main bronchus crosses the oesophagus. It is advisable for patients using this antibiotic to take the evening dose some time before retiring and with adequate volumes of fluid.

We thank Professor J M Evanson for permission to report this case.

¹ Collins FJ, Matthews HR, Baker SE, Strakova JM. Drug-induced oesophageal injury. *Br Med J* 1979;i:1673-6.

² Crowson TD, Head LH, Ferrante WA. Esophageal ulcers associated with tetracycline therapy. *JAMA* 1976;235:2747-8.

³ Evans KT, Roberts GM. Where do all the tablets go? *Lancet* 1976;iii:1237-9.

(Accepted 3 February 1981)

University Department of Medicine, Withington Hospital, Manchester M20 8LR

K S CHANNER, BSC, MRCP, senior house officer
D HOLLANDERS, MSC, MRCP, lecturer

Severe hypoglycaemia during physical exercise and treatment with beta-blockers

Despite important metabolic effects beta-blocking agents are usually well tolerated, but during metabolic stress important perturbations may occur. For instance, treatment with a non-selective beta-blocker leads to a slower rate of recovery from insulin-induced hypoglycaemia.¹ Similarly, blood glucose concentrations are more rapidly reduced during prolonged physical exercise during treatment with a beta-blocker.² Serious hypoglycaemia has occurred during prolonged exercise in patients receiving treatment with propranolol³ and pindolol.⁴

We report on a patient treated with alprenolol, a non-selective beta-blocker with intrinsic sympathomimetic activity, who developed serious hypoglycaemia during endurance exercise.

Case report

The patient was a previously healthy 61-year-old man who was accustomed to jogging for one to two hours several times a week. Hypertension had been detected six years previously and the treatment finally adjusted to alprenolol 100 mg twice daily and hydralazine 25 mg twice daily, with which his blood pressure was well regulated. He received no other medication and did not abuse alcohol. No diabetes was known in his family.

In 1979 he went cycling for about 1½-2 hours. He became increasingly fatigued and eventually lost consciousness. He recovered slowly on the way to a local hospital, where he was given glucose with immediate improvement.

Later in 1979 he went jogging after having had only a light lunch. After about 1-2 hours he felt increasing weakness and unsteadiness and then fell and could not rise. He was found unresponsive on the ground and brought to hospital. He had sinus bradycardia with a heart rate of around 30 beat/min. Blood pressure was 190/110 mm Hg. Blood glucose concentration was 1.9 mmol/l (34 mg/100 ml). He was given intravenous glucose with immediate improvement: he became orientated and gave adequate answers though could not remember what had happened after he had fallen. Heart rate stabilised around 60 beats/min and blood pressure around 160/90 mm Hg. Subsequent electrocardiography showed sinus rhythm with slightly raised ST intervals. Serum aspartate transaminase activity was slightly raised but

then normalised; serum alanine transaminase activity remained normal. Lactate dehydrogenase isoenzyme electrophoresis showed increases in isoenzymes of myocardial origin. These enzyme changes combined with the discrete electrocardiographic changes suggest the possibility of a myocardial infarction, which might have developed during the hypoglycaemia. He had not felt any chest pain while jogging.

Fasting blood glucose concentrations while he was in hospital were all in the normal range (around 4.2 mmol/l (76 mg/100 ml)) as was the fasting insulin concentration (3.0 mU/l).

He was subsequently discharged from hospital taking metoprolol (a cardioselective beta-blocking agent) 100 mg twice daily and hydralazine 25 mg twice daily. He was told to eat regularly, particularly when taking exercise. He remained well and resumed his previous level of physical activity.

Comment

In this patient one suspected and one verified episode of severe, symptomatic hypoglycaemia occurred in association with prolonged exercise. There was no evidence of an insulinoma or any other known cause of hypoglycaemia apart from the endurance exercise.

During prolonged, submaximal exercise glucose and free fatty acids are mainly taken up from the blood stream and used by the working muscles.⁵ Splanchnic glucose production cannot keep pace with the increased rate of use, leading to a fall in blood glucose concentrations.⁵ With beta-blockade, particularly with non-selective agents, this fall occurs more rapidly,² probably because of impaired glucose production in the liver and a greater dependency on glucose as substrate since the release of free fatty acids from the adipose tissue is impaired by these agents.¹

The haemodynamic pattern in our patient (bradycardia and raised blood pressure) was similar to that reported previously in patients with hypoglycaemia receiving concomitant treatment with a non-selective beta-blocker.¹ Thus a potentially serious reaction may occur during prolonged physical exercise and concomitant beta-blockade. Patients treated with beta-blockers should be particularly careful to ingest suitable energy-giving food when participating in endurance exercise.

¹ Lager I, Blohmé G, Smith U. Effect of cardioselective and non-selective beta-blockade on the hypoglycaemic response in insulin-dependent diabetics. *Lancet* 1979;i:458-62.

² Galbo H, Holst JJ, Christensen NJ, Hilsted J. Glucagon and plasma catecholamines during beta-receptor blockade in exercising man. *J Appl Physiol* 1976;40:855-63.

³ Aksnes EG. Beta-blokkere—farlig for skiløpere? *Tidsskrift for den Norske Laegeforening* 1977;97:576.

⁴ Uusitupa M, Aro A, Pietikäinen M. Severe hypoglycaemia caused by physical strain and pindolol therapy. *Ann Clin Res* 1980;12:25-7.

⁵ Wahren J, Felig P, Hagenfeldt L. Physical exercise and fuel homeostasis in diabetes mellitus. *Diabetologia* 1978;14:213-22.

(Accepted 9 February 1981)

Departments of Medicine I and II, Sahlgren's Hospital, University of Göteborg, Göteborg, Sweden

G HOLM, MD, associate professor of internal medicine

J HERLITZ, MD, resident

U SMITH, MD, associate professor of internal medicine

Educational value of printed information for patients with hypertension

Printed information booklets are widely used to inform patients about their condition. We investigated the educational value of one such booklet among patients with high blood pressure.

Patients, methods, and results

One hundred consecutive patients attending a blood pressure clinic were randomised into two groups, one of which received a booklet about hypertension¹ and the other attended the clinic as before. The booklet explained the reasons for controlling high blood pressure, the need for continuous treatment even in the absence of symptoms, the importance of cardiovascular